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MORTALITY IN SMALL ANIMALS EXPOSED IN A SHOCK TUBE TO SHARP-RISING OVERPRESSURES OF 3-4 MSEC DURATION

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MORTALITY IN SMALL ANIMALS EXPOSED IN A SHOCK TUBE TO "SHARP" RISING OVERPRESSURES OF 3-4 MSEC DURATION

FORWORD

This report describes research in the general area of blast and shock biology, more specifically the experiments deal with mortality and other biological consequences of exposure to "fast-rising" overpressures of 3-4 msec duration and the relation of these data to those in the literature dealing with the pressure-duration relationship as this effects lethality.

The data are limited to those circumstances in which exposure to single-pulse overpressures rising almost instantaneously occur. They are pertinent to military and industrial situations that involve explosive phenomena.

The present study is a part of a continuous program of research which has been under way since 1952 and future work will extend the interspecies investigations to the end that the human response following exposure to blast pressures will be better understood.

ABSTRACT

A total of 661 animals were exposed to "sharp"-rising overpressures of 3-4 msec duration using a shock tube of novel design which produced a pressure pulse similar to that obtained with high explosives. The reflected shock overpressures associated with 50 per cent lethality were 29.0, 38.6, 35.2 and 35.6 psi for the mouse, rat, guinea pig and rabbit respectively. Other observations included the time of death in mortally wounded animals and gross pathological lesions likely to contribute to mortality. Selected data from the literature bearing upon the influence of overpressure and pulse duration on lethality were reviewed. These included pulse durations ranging from less than 1 msec to 6-8 sec. The critical pulse duration, that duration shorter than which the overpressures required for mortality increases sharply, was noted to depend upon animal size and to be of the order of many hundreds of microseconds to very few milliseconds for "smaller" animals and a few to many tens of milliseconds for "larger" animals.

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INTRODUCTION

As pointed out in previous studies[10-13,18-21] quantitative tolerance of man to blast-produced overpressures is difficult to estimate precisely. However, information now available from considerable animal experimentation makes it apparent that the mammal is extraordinarily sensitive to the rate, character and magnitude of the pressure rise, the duration of the pulse, and to a lesser extent to the rate, character and magnitude of the pressure fall. Too, it is known that these factors vary not only with explosive yield, range and burst conditions, but also are modified by the geometry in which exposure occurs. Consequently, interpretation of earlier and more recent biological data must be guided by careful attention to the physical details of the environmental pressure-time variation with which a given biological effect is associated if adequate understanding is to be forthcoming and apparent conflicts in data are to be clarified. For example, mortality data were obtained in 1941 by English workers[8] using 5 species of animals exposed in air to "fast"-rising, "short"-duration overpressures produced by detonation of small (1, 8 and 66.6 lb) high-explosive charges. By extrapolation of these data, 50 per cent lethal conditions for the 60 and 80 kg animal were predicted to be near 390 and 470 psi, respectively.[8] Field studies carried out during the first World War, also in England, noted 12 human exposures for which the estimated high-explosive produced, maximal overpressures ranged from 170 to 500-600 psi with only one fatality related to an estimated overpressure of 450 psi.[8]

In contrast, experimentation with larger high-explosive charges (55 to 4000 lbs) in the early 1940's in Germany revealed that lethal overpressures for dogs dropped from near 218 psi for a "fast"-rising overpressure enduring for 1.6 msec to 76 psi when the pulse duration was 11.8 msec.[5] Field studies in Germany involving exposure of 13 individuals – located in a gun revetment – to blast from a 2000 lb bomb (1210 lbs of explosive), reported two fatalities at estimated maximal overpressures of 235 psi, which pressure resulted from a reflection of an incident wave of 58 psi. The duration of the pulse was between 4 and 6 msec.

Also, in apparent contradiction to the above-noted British and German

results, are recent shock-tube data obtained on 6 species of animals subjected to "fast"-rising overpressures of near 400 msec duration which when extrapolated to the 70 kg animal allowed the 50 per cent lethal overpressure to be calculated at 50.5 psi.[13] These findings led to a tentative, recent prediction of 45 to 55 psi as the overpressure range for 50 per cent lethality in humans exposed to "sharp"-rising, nuclear-produced overpressures of "long" duration.[21]

Actually, there is no conflict among the British, German and American data cited above. The differences are simply more apparent than real, there being ample evidence that (a) both the magnitude and duration of "fast"-rising, single-pulse overpressures must be considered in specifying tolerance to "shorter" duration overpressures as has been pointed out by German,[5, 16] English,[17] Swedish,[3, 4] and American workers[9-13, 18-21] independently; (b) the character, rate and magnitude of the pressure rise not only can be markedly influenced by the circumstances of exposure, but also are sensitive to other factors which can make the free-field wave form atypical, all facts that are often critical for many types of field data; and (c) the overpressure duration is mostly determined by total yield and range which for overpressures near 100 psi, even for yields as low as 10 kilotons, is near 200 msec for sea-level detonations in air. Such findings not only allow integration of the earlier fragmentary animal experiments employing "short"-duration, high-explosive produced overpressures with the more recent, but as yet incomplete, observations employing "long"-duration pulses obtained full-scale and in the laboratory, but also focuses attention, among other things, on the need for systematic investigation of the parameter of overpressure duration.

The experiments to be reported here are a part of a broad study primarily conceived to establish an interspecies correlation between the weight of animals and their tolerance to "sharp"-rising overpressures as a function of pulse duration. However, the limited objective of the present report is to set forth the empirically determined relationship between lethality and the magnitude of single, "sharp"-rising overpressures of 3-4 msec duration for mice, rats, guinea pigs and rabbits, and to record selected, but significant, gross pathological lesions caused by air blast generated in a shock tube specially designed to produce pressure pulses similar to "small", high-explosive charges.

METHODS

Geometry of Shock Tube.

A diagram of the shock tube developed to produce steep-fronted pressure waves of "short" duration appears in Fig. 1. The apparatus consisted of a cylindrical compression chamber one foot in length separated from the expansion chamber by a rupturable diaphragm. The expansion chamber was composed of a 15-ft length of steel tubing with a test section bolted to its downstream end. The latter was shaped like a mathematical plus symbol (+). As noted in Fig. 1, the lateral members of the test section remained open and provided vents of 23-1/2 in. in diameter. The distal end was closed by a steel plate (the end-plate). The shock tube was circular in cross section with an internal diameter of 23-1/2 in. Its wall thickness was 3/8 in.

The diaphragm consisted of one or more sheets of plastic film*, 0.010 or 0.0075 in. thickness. Each sheet measured 34 x 34 in. and had holes pre-drilled to match those of the flanges. For their installation they were simply bolted between the two flanges of the diaphragm station.

Diaphragm rupture was initiated by a .22 caliber bullet fired from a pistol mounted on the outside wall of the tube just downstream from the diaphragm station. Diaphragms that consisted of more than three sheets of plastic were ruptured by a 12-gauge shotgun instead of the .22 caliber pistol.

Air was pumped into the compression chamber to a pre-determined pressure. Following diaphragm rupture, the compressed air that escaped from the compression chamber generated a shock wave which traveled down the expansion chamber and reflected from the plate closing the end of the tube. Simultaneously, a rarefaction, or negative wave, was produced at diaphragm rupture; it traveled back into the compression chamber, reflected from the back end of that chamber, and moved downstream, markedly reducing the pressure of the gas through which it passed. Because it was traveling at a velocity greater than that of the incident shock front, it eventually overtook and weakened the shock; this occurred at a distance downstream equal to about 16 times the length of the compression chamber.

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*DuPont MYLAR, a polyester film.

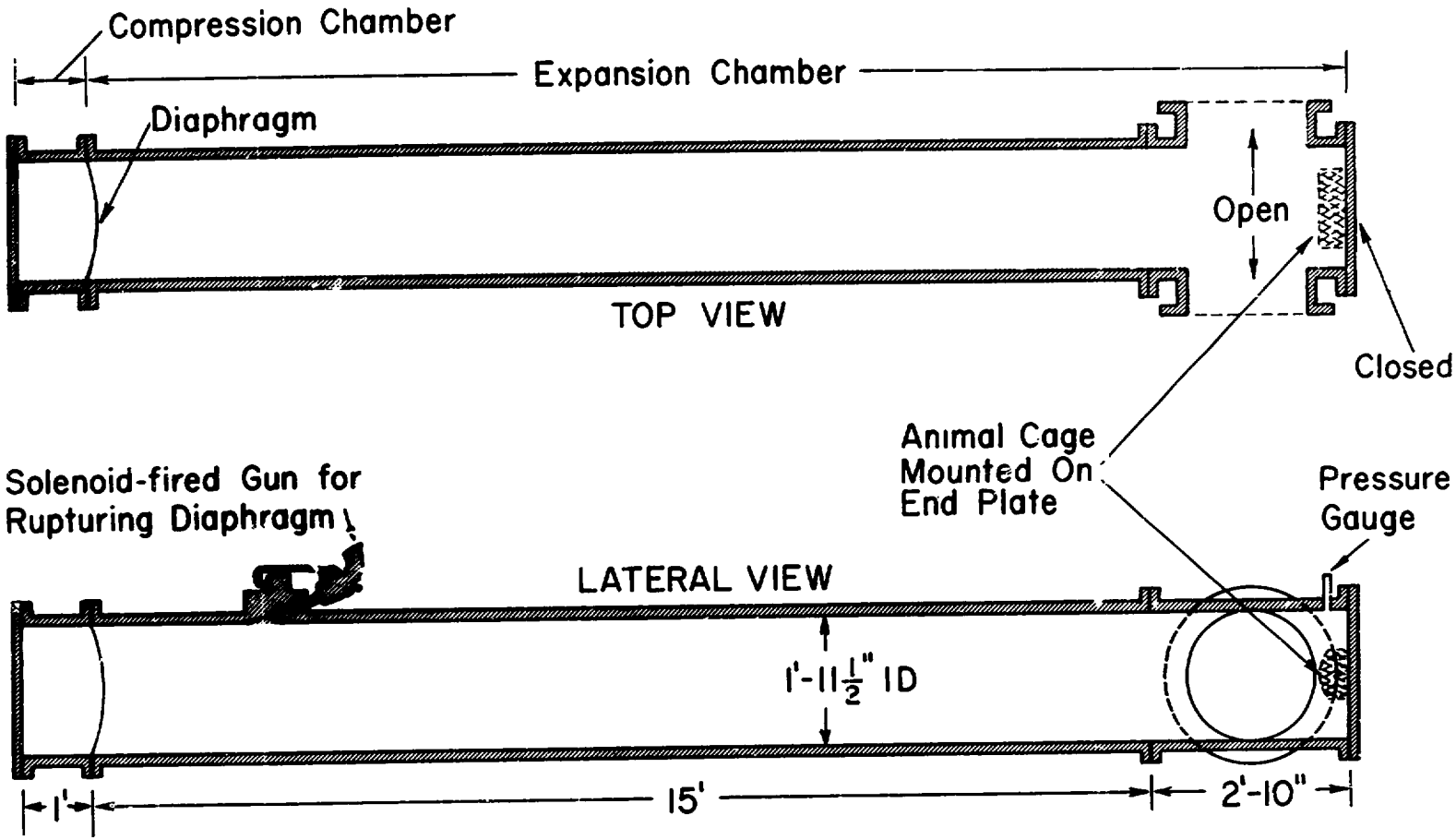

Compression Chamber
Expansion Chamber
Diaphragm
Open
TOP VIEW
Closed
Animal Cage
Mounted On
End Plate
Pressure
Gauge
Solenoid-fired Gun for
Rupturing Diaphragm
LATERAL VIEW
1'-11½" ID
1'
15'
2'-10"

Figure 1

Since the downstream end of the expansion chamber was closed, the shock front, upon striking the end-plate, reflected and travelled back upstream creating a region of high, stagnant pressure. The pressure behind the reflected shock front was more than double that following the incident shock. Consequently, by opening the lateral ports of the "Test" section, the duration of the high reflected pressure obtained at the end-plate was markedly reduced by the rarefaction waves from the compression chamber and the lateral openings. For a detailed account of shock tube theory, the reader is referred elsewhere.[2]

Pressure-time Measurements

The pressure-time history in the test section was measured and recorded by commercially available electronic equipment. On each test the pressure was measured by a quartz piezoelectric transducer (SLM PZ-14)* It was shock-mounted flush with the wall of the tube at a point 3 in. from the end-plate. The signal from the gauge was fed through low noise cable to an amplified-calibrator (Model 455A)* and then into an oscilloscope (Tektronix, Model 535). A permanent record was obtained by photographing the sweep on the oscilloscope with a Polaroid Land Type Camera.

The quartz piezoelectric gauge was statically calibrated by using a small air pressurization tank. Its dynamic performance was periodically checked on a calibration shock tube.

Experimental Animals

A total of 661 young adult animals was utilized in this study, of which there were 240 mice, 160 rats, 177 guinea pigs, and 84 rabbits.

All animals, one to a cage, were exposed — always with the right side upstream — in steel diamond-mesh cages that were bolted against the inside surface of the end-plate. Table 1 lists the pertinent animal and cage information. Photographs of animals in their respective cages are shown in Fig. 2.

Since the test section was open laterally, the animals were observed immediately after each shot. Animals were followed for a two-hour postshot period. Deaths were recorded at the end of each successive minute for the first 20 min

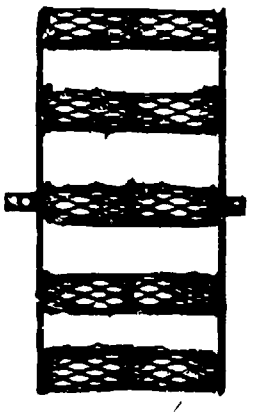
*Purchased from Kistler Instrument Co., North Tonawanda, New York.

Table 1

Number, Species, Weight and
Age of Animals and Dimensions of Exposure Cages

Species and Type of Animals	Sex and number of animals	Body wt. (gm) Average & range	Body wt. (gm) S. D.	Age (months)	Dimensions of individual cage (in.)*	Number of compartments
Mouse Webster strain	(F) 240	20.9 (14.4-26.7)	± 6.2	1 to 1-1/2	1-1/2x1-1/2x3-1/4	10
Rat Sprague Dawley	(F) 160	183 (156-218)	± 12.6	2 to 2-1/2	2x2-1/4x8	5
Guinea pig English breed	(M&F) 177	436 (363-577)	± 39.3	3-1/2 to 4	3x3x8-1/2	3
Rabbit New Zealand White	(F) 84	1810 (1190-2722)	±293.3	2-1/2 to 3	5x5x14	2
Total	661					

*See Figure 2 for photographs of animals in cages.



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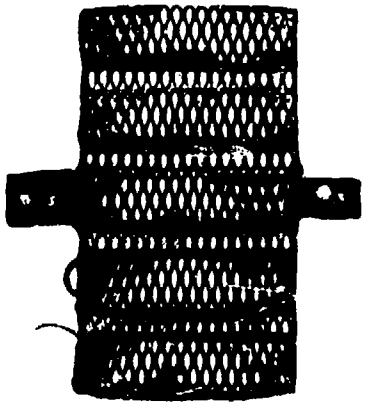

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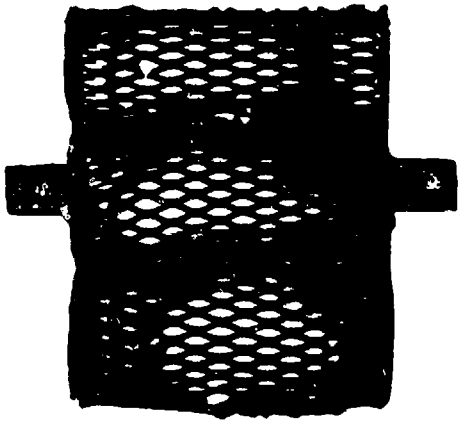

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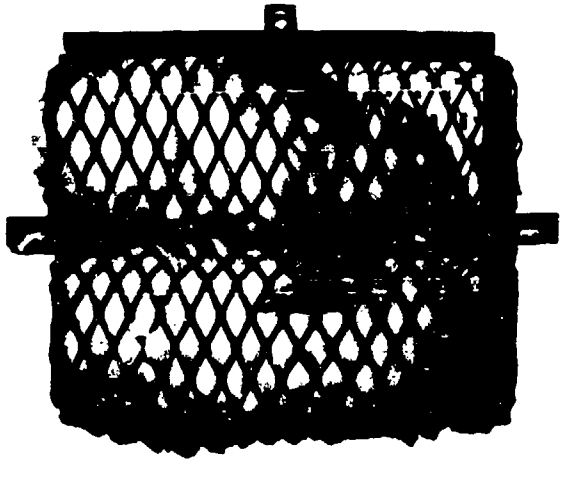


(d)

Figure 2

and then every 5 min for the next 2 hours. Survivors were sacrificed and autopsied after 2 hours; those killed by the blast were autopsied as soon as possible after death, usually within 15 min.

RESULTS

Pressure-time History

Characteristic pressure-time records taken with the gauge, side-on, 3 in. from the end-plate and face-on, flush with the inside of the end-plate are illustrated in Fig. 3. The maximal reflected shock pressures and the duration of the overpressures measured with the gauges at 3 in. and at the end-plate were in good agreement. Figure 4 gives the average incident and reflected shock pressures obtained over the range of compression-chamber pressures employed. The durations of these overpressures ranged between 3 and 4 msec. Because there was appreciable variation from the average data shown in Fig. 4, it was necessary to measure the pressure data each time animals were exposed.

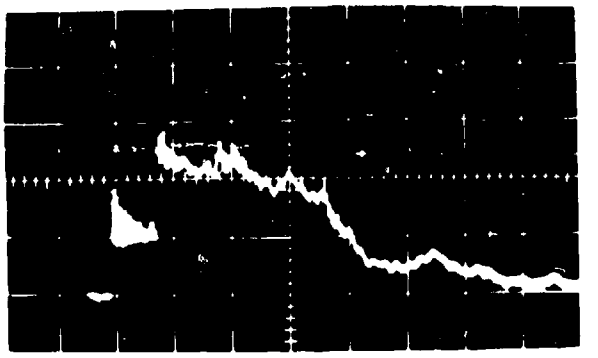
Mortality as Related to Reflected Pressure

The pressure-time data, metered for each shot, along with the corresponding mortality for each species were listed according to the ascending order of the reflected shock pressures; the figures were then grouped and the averages assembled as in Tables 2 to 5. The mortality observations represent lethality in 2 hours.

The probit analysis of Finney[6] was applied to the mortality data in Tables 2 through 5; the procedure was programmed for a Bendix G-15 Computer which accomplished all the calculations to provide probit regression line equations relating percent mortality in probit units to the log of the reflected (maximal) pressure. The results of the probit analysis are plotted in Fig. 5 and are summarized in Table 6.

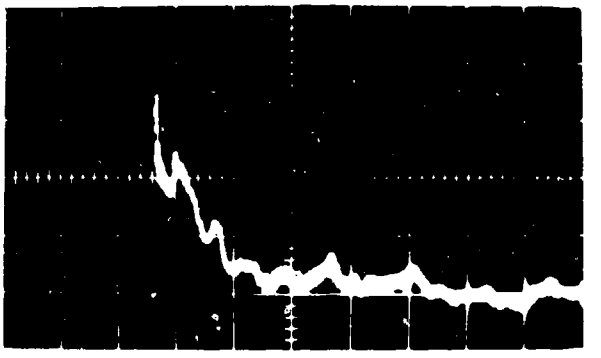
Substituting values of $y = 5$ (50 per cent mortality) into the respective probit equation and solving for x yielded the maximal or reflected pressure necessary for 50 per cent lethality, the LD_{50}. For each species results were as follows: mouse, 29.0 psi; rat, 38.6 psi; guinea pig, 35.2 psi; and rabbit, 35.6 psi (Table 6 and Fig. 5). Statistical tests showed the mouse LD_{50} to be signif-

QUARTZ GAUGE PRESSURE-TIME RECORDS



0.5 msec/cm

Side-on 3" from end plate



2.0 msec/cm

Face-on at the end plate

Figure 3

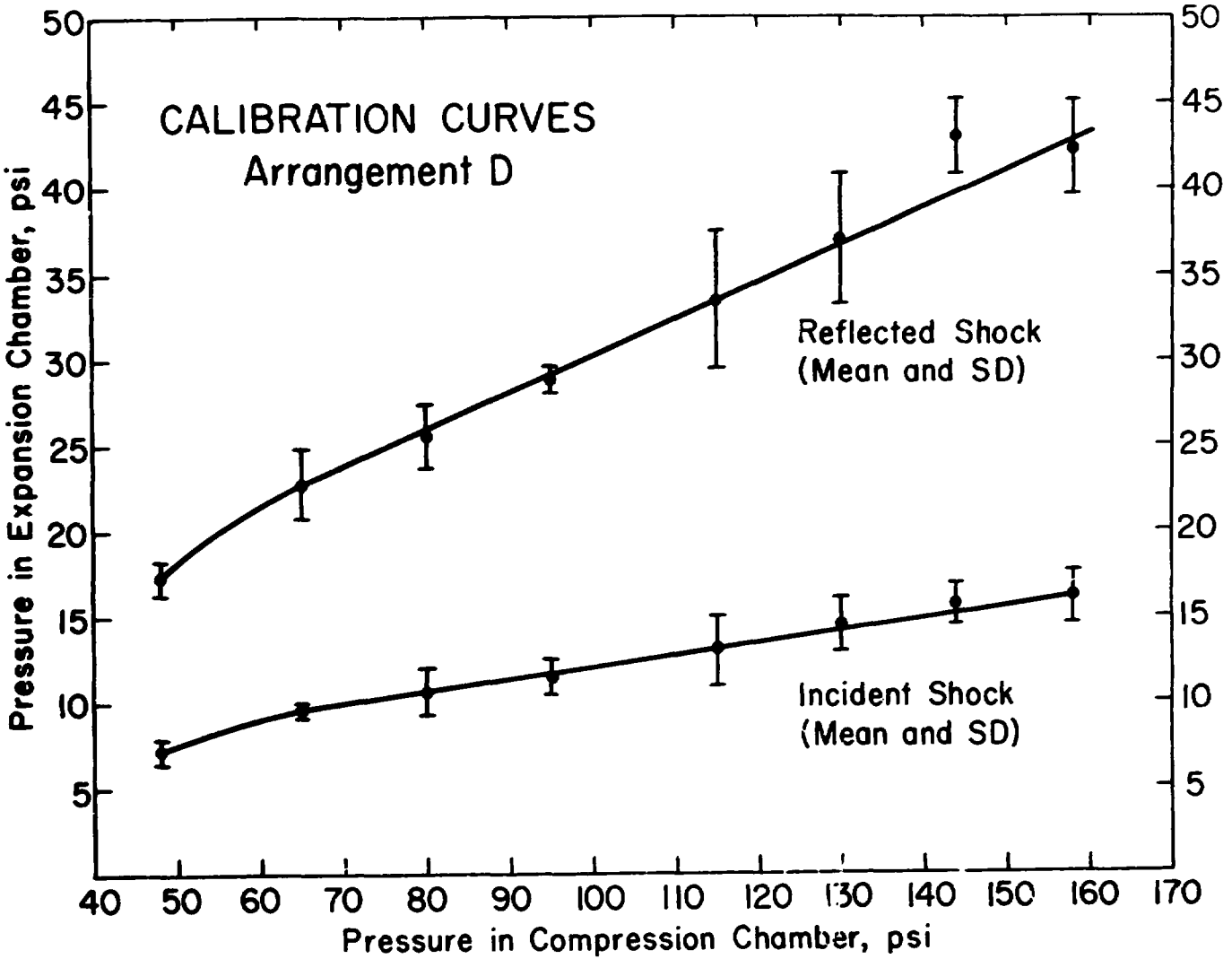

CALIBRATION CURVES
Arrangement D
Reflected Shock
(Mean and SD)
Incident Shock
(Mean and SD)
Pressure in Expansion Chamber, psi
Pressure in Compression Chamber, psi
50
45
40
35
30
25
20
15
10
5
40
50
60
70
80
90
100
110
120
130
140
150
160
170

Figure 4

Table 2

Mouse Mortality Data

Group	Overpressure (psi)		Mortality	
	Incident Shock	Reflected Shock	Dead	Percent
	Average and Range	Average and Range	Total	
1	3.2 (2.7-3.4)	7.6 (6.8-8.0)	0/30	0
2	6.8 (6.4-7.3)	16.2 (15.0-18.2)	0/30	0
3	9.3 (8.2-10.0)	22.8 (21.4-23.6)	3/40	7.5
4	11.4 (10.9-11.8)	24.8 (24.1-25.5)	2/20	10.0
5	11.8 (10.9-12.7)	29.7 (29.0-30.9)	19/30	63.3
6	14.5 (14.5)	35.5 (34.5-36.4)	20/20	100.0
7	15.2 (13.6-16.4)	39.1 (39.1)	27/30	90.0
8	16.1 (15.5-16.4)	40.3 (40.0-40.9)	29/30	96.7
9	17.3 (-)	45.5 (-)	10/10	100.0
Total			110/240	
			Computed LD_{50} = 29.0 psi	

Table 3

Rat Mortality Data

Group	Overpressure (psi)		Mortality	
	Incident Shock Average and Range	Reflected Shock Average and Range	Dead Total	Percent
1	7.7 (7.7)	18.6 (18.6)	0/10	0
2	11.9 (10.9-12.7)	30.8 (29.1-32.7)	0/25	0
3	14.7 (14.1-15.4)	36.4 (35.9-36.8)	3/35	8.6
4	15.4 (14.5-17.1)	38.0 (37.7-38.2)	17/30	56.7
5	16.7 (14.5-20.8)	39.3 (38.8-39.5)	15/20	75.0
6	17.0 (15.2-18.3)	41.8 (40.8-43.2)	21/25	84.0
7	19.9 (17.3-21.7)	46.5 (44.5-48.3)	14/15	93.3
Total			70/160	
		Computed LD_{50} = 38.6 psi		

Table 4

Guinea Pig Mortality Data

Group	Overpressure (psi)		Mortality	
	Incident Shock Average and Range	Reflected Shock Average and Range	Dead Total	Percent
1	3.6 (3.6)	9.2 (9.1-9 5)	0/9	0
2	7.3 (6.4-8.2)	17.5 (15.9-19.5)	0/18	0
3	9.3 (-)	22.4 (20.9-25.9)	0/15	0
4	10.6 (9.1-11.4)	27.7 (26.4-28.6)	4/21	19.0
5	12.0 (10.9-13.6)	32.4 (30.5-35.5)	8/33	24.2
6	14.7 (12.7-15.5)	37.3 (36.4-39.1)	13/24	54.2
7	15.0 (13.6-16.4)	41.5 (40.0-43.6)	38/45	84.4
8	16.4 (16.4)	45.6 (45.2-46.4)	12/12	100.0
Total			75/177	
			Computed LD_{50} = 35.2 psi	

Table 5

Rabbit Mortality Data

Group	Overpressure (psi)		Mortality	
	Incident Shock	Reflected Shock	Dead	Percent
	Average and Range	Average and Range	Total	
1	3.6 (3.6)	6.4 (6.4)	0/2	0
2	7.0 (6.4-7.3)	17.7 (16.8-18.6)	0/8	0
3	10.1 (9.1-12.7)	23.5 (22.7-24.0)	0/8	0
4	10.6 (10.0-10.9)	27.4 (26.4-29.1)	0/8	0
5	11.7 (10.9-12.7)	30.7 (30.0-30.9)	2/14	14.3
6	13.2 (11.8-16.4)	34.5 (32.7-35.5)	6/14	42.9
7	14.6 (14.5-15.0)	37.8 (36.4-39.1)	7/12	58.3
8	15.6 (14.5-18.2)	41.2 (40.0-42.7)	11/12	91.7
9	16.1 (14.5-17.3)	45.6 (45.0-46.4)	6/6	100.0
Total			32/84	
			Computed LD_{50} = 35.6 psi	

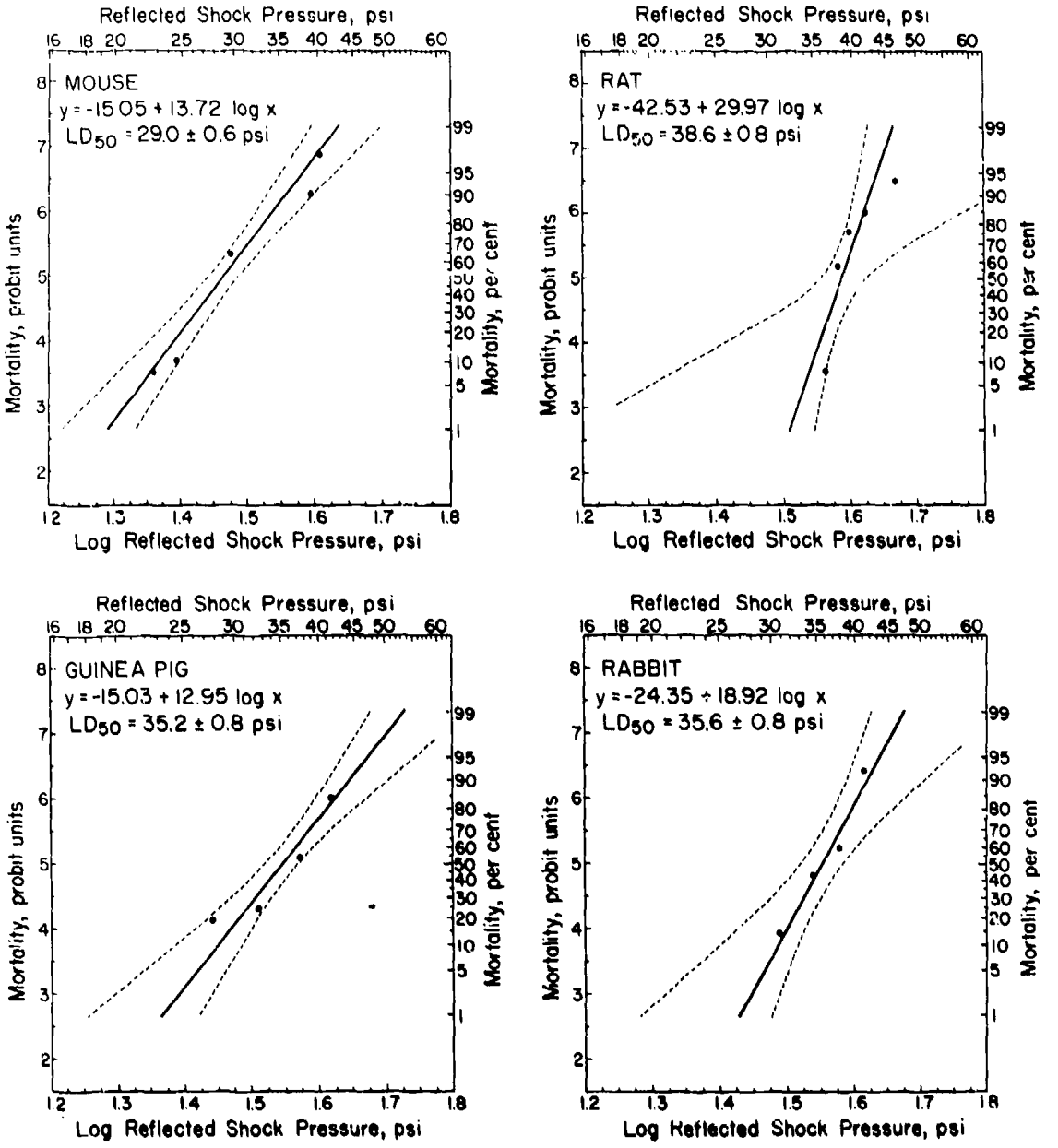

Reflected Shock Pressure, psi
16 18 20 25 30 35 40 45 50 60
MOUSE
y = -15.05 + 13.72 log x
LD50 = 29.0 ± 0.6 psi
RAT
y = -42.53 + 29.97 log x
LD50 = 38.6 ± 0.8 psi
GUINEA PIG
y = -15.03 + 12.95 log x
LD50 = 35.2 ± 0.8 psi
RABBIT
y = -24.35 + 18.92 log x
LD50 = 35.6 ± 0.8 psi
Mortality, probit units
Mortality, per cent
Log Reflected Shock Pressure, psi

Figure 5

Table 6

Summary of the Probit Analysis

Species	LD_{50} reflected pressure psi	Standard error of the LD_{50} psi	Probit regression equation constants		
			a, intercept	b, slope	s(B)*
Mouse	29.0	±0.6	-15.05	13.72	1.536
Rat	38.6	±0.8	-42.53	29.97	9.368
Guinea pig	35.2	±0.8	-15.03	12.95	1.903
Rabbit	35.6	±0.8	-24.35	18.92	3.908

*Standard error of the slope constant.

icantly lower than the other species at the 95 per cent confidence level. That for the rat was significantly higher than the guinea pig's, also at the 95 per cent confidence level, but not higher than the rabbit's. The guinea pig and the rabbit LD_{50}'s were not significantly different.

Statistical tests further revealed that the slopes of the probit mortality curves of the mouse, guinea pig, and rabbit were parallel at the 95 per cent confidence interval. The slope of the rat curve was not "statistically" parallel with the slopes for the other three species.

Gross Pathological Findings

Time of death. Table 7 summarizes the number of deaths that occurred over the 2-hr postshot period by 5 min intervals. As seen in the table, 187 animals (65 per cent) of the total 287 expired within 5 min. By the end of 30 min and 60 min, 93 and 96 per cent, respectively, had died. There were but 3 mice, 4 guinea pigs, and 3 rabbits dying between 1 and 2 hrs. Consequently, there was little difference between the LD_{50}'s calculated on the basis of 1 or 2 hr mortality.

The data are plotted in Fig. 6 to show the cumulative percent mortality as a function of time. As noted in the figure, the same pattern generally held for each species; namely, the mortality rose rapidly over the first 5 min, less rapidly for the next 25 to 30 min, and slowly during the remaining hour and a half. On the average, there was a tendency for the mean survival time to be shorter the higher the overpressure of exposure.

Nasal and oral signs. Usually, the only external sign visually detectable in fatalities, other than cessation of breathing often preceded by a gasping respiration, was the appearance of a pink or red-tinged froth at the nares and/or the lips. Occasionally frank bleeding was observed, mostly of short duration. The source of blood was from the pulmonary tree and not the nasal membranes, the sinuses or the pharynx. Table 8 lists the number of surviving and mortally wounded animals showing these nasal and oral signs. It is clear that the appearance of blood-tinged froth or bleeding from the nose and/or mouth was a grave sign; no surviving animal exhibited this picture, while 63 per cent of the fatalities did. Even so, there was a sharp interspecies difference as the last

Table 7

Lethality as Related to Time

Time Period (min)	Number of deaths within indicated time periods					Per cent of total No. dead	Cumulative per cent
	Mouse	Rat	Guinea pig	Rabbit	Total		
0-5	93	40	38	16	187	65	65
6-10	7	7	11	4	29	10	75
11-15	1	4	9	6	20	7	82
16-20	4	8	7	0	19	7	89
21-25	0	4	2	2	8	3	92
26-30	0	5	0	0	5	2	93
31-35	0	0	1	0	1	0.3	94
36-40	1	0	0	1	2	0.7	94
41-45	0	2	2	0	4	1	96
46-50	0	0	1	0	1	0.3	96
51-55	1	0	0	0	1	0.3	96
56-60	0	0	0	0	0	0	96
61-65	0	0	0	0	0	0	96
66-70	0	0	0	0	0	0	96
71-75	1	0	1	1	3	1	98
76-80	0	0	0	0	0	0	98
81-85	0	0	0	0	0	0	98
86-90	1	0	2	0	3	1	99
91-95	0	0	0	0	0	0	99
96-100	0	0	0	0	0	0	99
101-105	0	0	1	0	1	0.3	99
106-110	1	0	0	1	2	0.7	100
111-115	0	0	0	1	1	0.3	100
116-120	0	0	0	0	0	0	100
Total No. Dead	110	70	75	32	287		

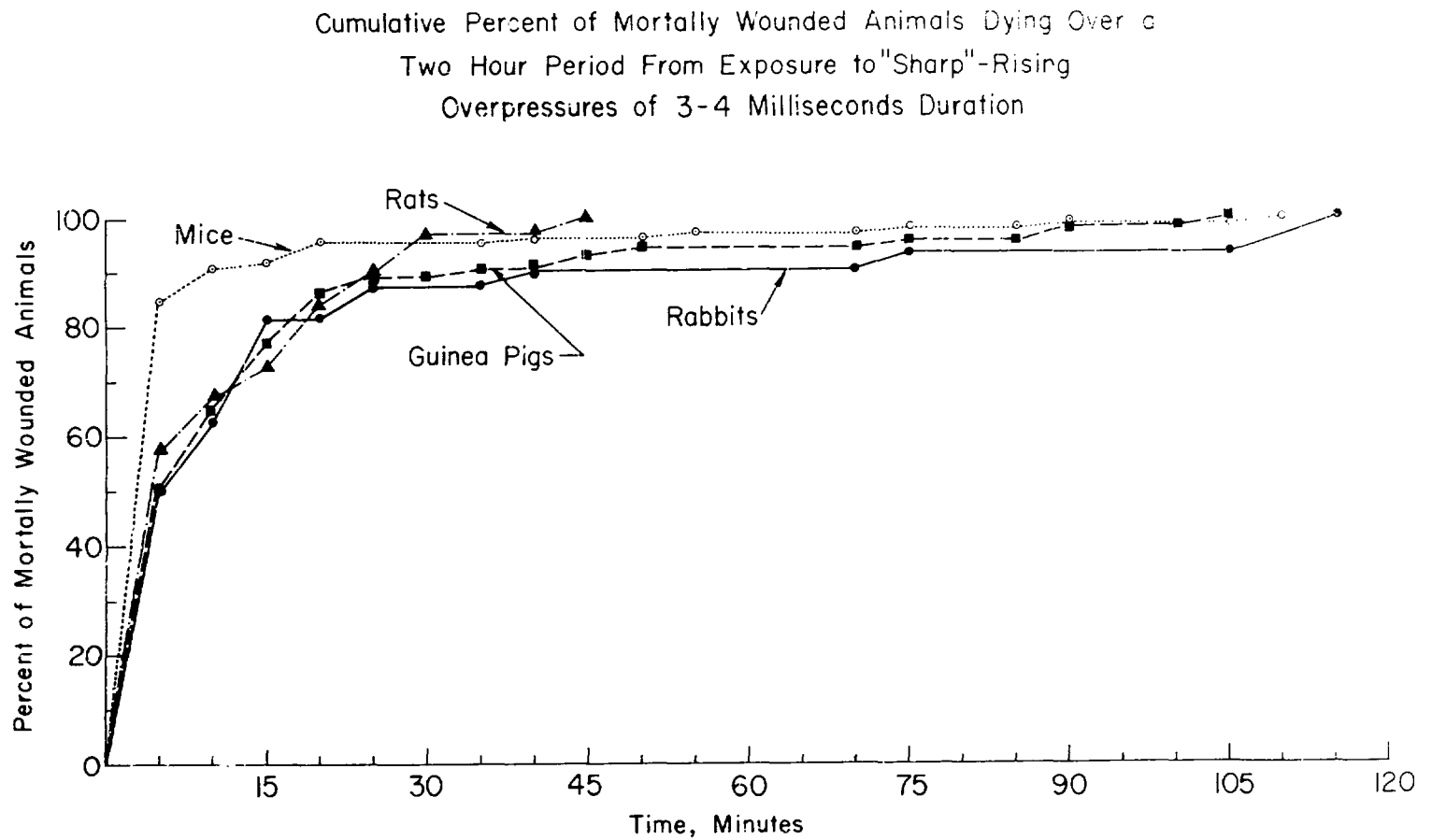

Cumulative Percent of Mortally Wounded Animals Dying Over a
Two Hour Period From Exposure to "Sharp"-Rising
Overpressures of 3-4 Milliseconds Duration
Rats
Mice
Rabbits
Guinea Pigs
Percent of Mortally Wounded Animals
0
20
40
60
80
100
15
30
45
60
75
90
105
120
Time, Minutes

Figure 6

Table 8

Number Animals with Bloody Froth or Frank Bleeding
From Nose and/or Mouth

Species	Survivors			Fatalities		
	Number animals	No. with nasal and oral signs	%	Number animals	No. with nasal and oral signs	%
Mice	130	0	0	110	101	91.8
Rats	90	0	0	70	50	71.4
Guinea pigs	102	0	0	75	14	18.7
Rabbits	52	0	0	32	17	53.1
Total	374	0	0	287	182	63.4

column of Table 8 details and many animals died without any external evidence of hemorrhage whatsoever.

Lung weights. As expected, the lungs were severely damaged by the air blast. Tables 9 through 12 give the lung weights as a per cent of the body weight for each species grouped to show the relation between the different pressures of exposure and the mean lung weights for mortally wounded and surviving animals. The increase in lung weights over controls can be taken as an index of the amount of blood and edema fluid present in the lung as a result of exposure to overpressure. Also, this increase in lung weight can be roughly correlated with the severity of blast, as set forth graphically in Figures 7 through 10.

The figures show that the lung weights of exposed animals increased rapidly with increasing overpressure and then tended to level off. In general, the lungs of fatally injured animals were, on the average, heavier than the survivors in the corresponding pressure group. The instances in which the percent lung weights for surviving and dead animals were significantly different at the 95 per cent confidence level are indicated in Tables 9 through 12. It can be seen that the difference was significant for those pressure groups near the LD_{50} if adequate numbers of animals were exposed. At the higher and lower overpressures the number of animals surviving and dying, respectively, were too small to allow adequate statistical comparison.

The variations in lung weight as a function of time of death is of interest. Since the data for all species were similar, only those for guinea pigs are shown here in Fig. 11. That there was the greatest variation in the lung weights for animals dying within about 5 min is evident. To the contrary, those succumbing later exhibited less spread in the figures. The reasons for the findings are not revealed by the present study, but it is evident that a satisfactory view of the etiology of blast-produced lung damage and mortality must recognize these facts.

In the course of the mortality experiments some animals were exposed to sublethal pressures and the occurrence of lung hemorrhage was incidentally noted. Tables 9 through 12 give the number of animals showing this lesion when it occurred in the low-pressure groups. While the results do not allow

Table 9

Mouse Lung Weights

Group number	Reflected pressure psi	Lung weights as percent of body weight: Survivors Number of cases	Survivors Lung weight	Fatalities Number of cases	Fatalities Lung weight
1	7.6	30*	1.00±0.17**	0	- -
2	16.2	30	1.06±0.21	0	- -
3	22.8	37	1.29±0.22	3	1.81±0.37'
4	24.8	18	1.54±0.23	2	1.86±0.35
5	29.7	10	1.46±0.32	19	1.80±0.30'
6	35.5	0	- -	20	1.98±0.27
7	39.1	3	1.62±0.48	27	1.63±0.25
8	40.3	1	1.31 -	29	1.71±0.25
9	45.5	0	- -	10	1.67±0.26

* There were 17 mice in this group that sustained a slight degree of pulmonary hemorrhage.

** The mean and standard deviation.

' Indicates the mean was significantly higher than that of the corresponding survival group at the 95 percent confidence level.

The mean percent lung weight and standard deviation for 100 control mice was 0.88 ± 0.03.

Table 10

Rat Lung Weights

Group number	Reflected pressure psi	Lung weights as percent of body weight			
		Survivors		Fatalities	
		Number of cases	Lung weight	Number of cases	Lung weight
1	18.6	10*	0.65±0.12**	0	
2	30.8	25	1.05±0.29	0	
3	36.4	32	1.35±0.33	3	1.95±0.09'
4	38.0	13	1.58±0.24	17	2.25±0.45'
5	39.3	5	1.67±0.48	15	2.21±0.35'
6	41.8	4	1.52±0.26	21	2.23±0.37'
7	46.5	1	1.77 -	14	2.30±0.34

*Eight of these ten animals sustained a slight degree of pulmonary hemorrhage.

**The mean and standard deviation.

' Indicates the mean was significantly higher than that of the corresponding survival group.

The mean percent lung weight and standard deviation for 75 control rats was 0.71±0.09.

Table 11

Guinea Pig Lung Weights

Group number	Reflected pressure psi	Lung weights as percent of body weight			
		Survivors		Fatalities	
		Number of cases	Lung weight	Number of cases	Lung weight
1	9.2	9	0.70±0.06*	0	
2	17.5	18**	0.68±0.12	0	
3	22.4	15	0.99±0.33	0	
4	27.7	17	1.09±0.27	4	1.85±0.40'
5	32.4	25	1.32±0.45	8	1.86±0.52'
6	37.3	11	1.55±0.37	13	1.98±0.32'
7	41.5	7	1.90±0.48	38	2.30±0.55
8	45.6	0	- -	12	1.92±0.39

*The mean and standard deviation.

**Five of these 18 animals sustained a slight degree of pulmonary hemorrhage.

' Indicates the mean was statistically higher than that of the corresponding survival group.

The mean percent lung weight and standard deviation for 31 control guinea pigs was 0.83±0.15.

Table 12

Rabbit Lung Weights

Group number	Reflected pressure psi	Lung weights as percent of body weight			
		Survivors		Fatalities	
		Number of cases	Lung weight	Number of cases	Lung weight
1	6.4	2	0.91±0.24*	0	
2	17.7	8**	0.62±0.12	0	
3	23.5	8	0.86±0.38	0	
4	27.4	8	0.87±0.41	0	
5	30.7	12	1.16±0.22	2	1.25±0.02
6	34.5	8	1.30±0.36	6	1.62±0.17
7	37.8	5	1.15±0.17	7	1.55±0.21'
8	41.2	1	1.22 -	11	1.50±0.15
9	45.6	0	- -	6	1.56±0.31

*The mean and standard deviation.

**Seven of these eight animals sustained a slight degree of pulmonary hemorrhage.

' Indicates the mean was significantly higher than that of the corresponding survival group.

The mean percent lung weight and standard deviation for 46 control rabbits was 0.54 ± 0.13.

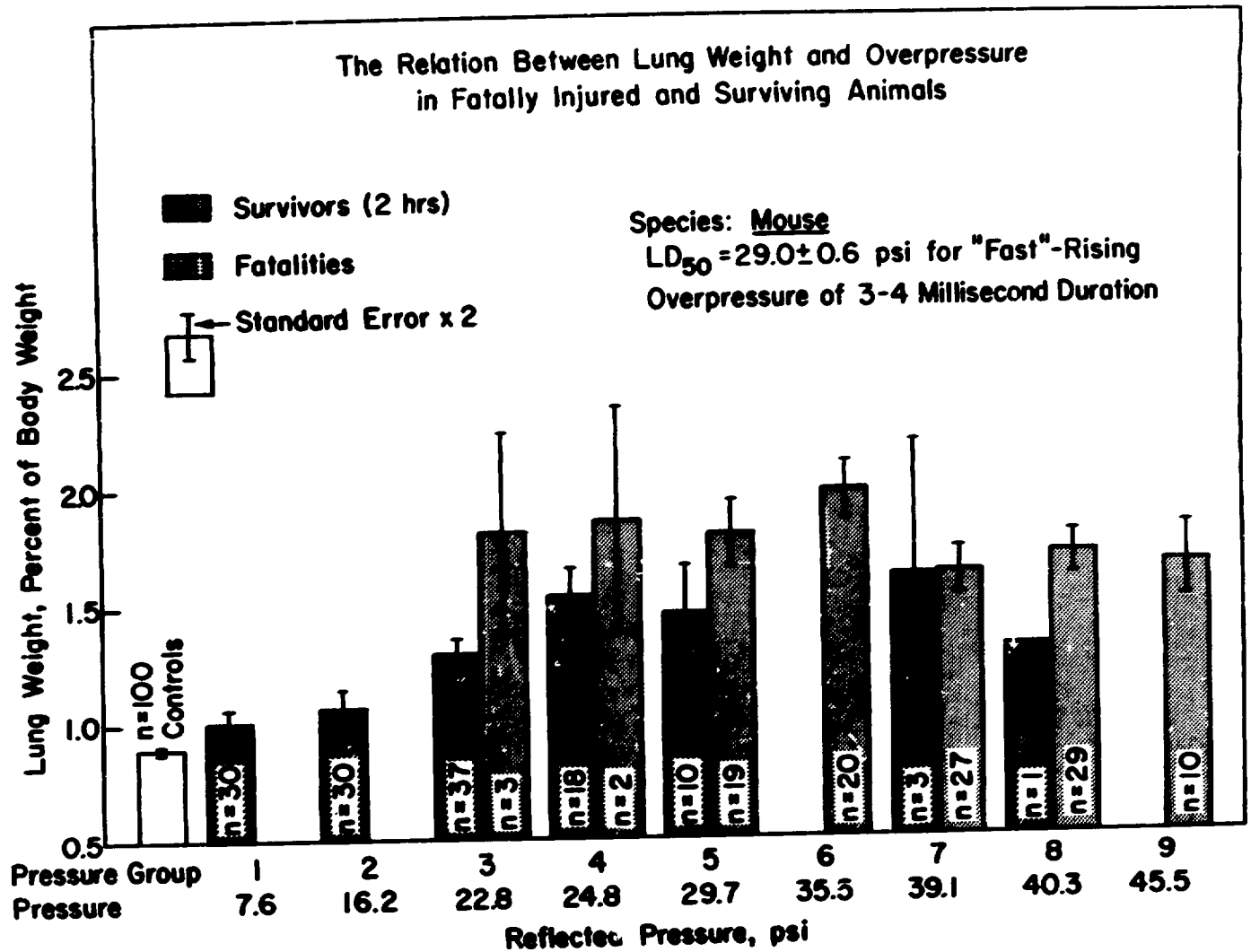

The Relation Between Lung Weight and Overpressure
in Fatally Injured and Surviving Animals
Survivors (2 hrs)
Fatalities
Standard Error x 2
Species: Mouse
LD50 = 29.0 ± 0.6 psi for "Fast"-Rising
Overpressure of 3-4 Millisecond Duration
Lung Weight, Percent of Body Weight
0.5
1.0
1.5
2.0
2.5
n=100 Controls
n=30
n=30
n=37
n=3
n=18
n=2
n=10
n=19
n=20
n=3
n=27
n=1
n=29
n=10
Pressure Group
Pressure
1
7.6
2
16.2
3
22.8
4
24.8
5
29.7
6
35.3
7
39.1
8
40.3
9
45.5
Reflected Pressure, psi

Figure 7

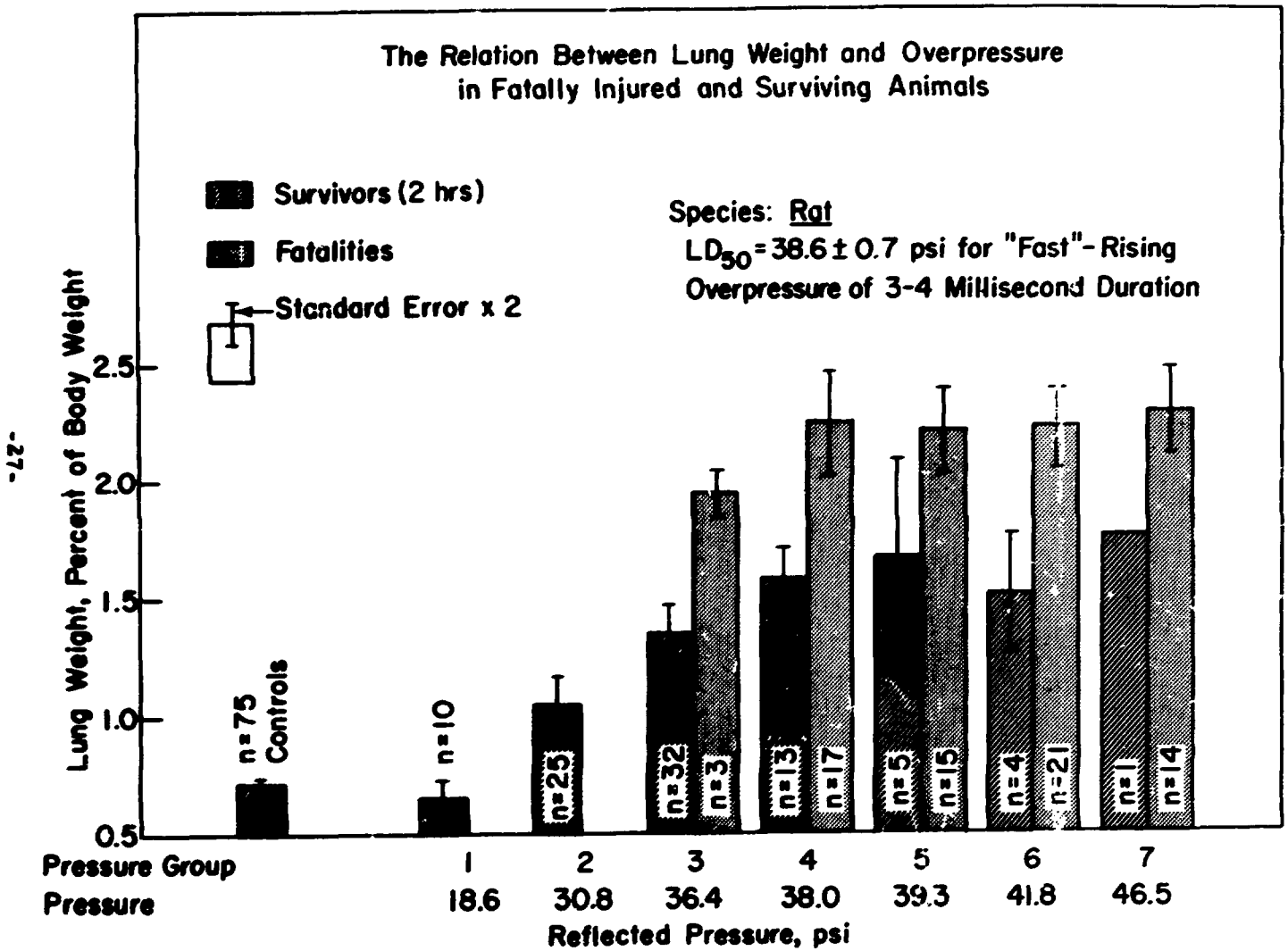

The Relation Between Lung Weight and Overpressure
in Fatally Injured and Surviving Animals
Survivors (2 hrs)
Fatalities
Standard Error x 2
Species: Rat
LD50 = 38.6 ± 0.7 psi for "Fast"-Rising
Overpressure of 3-4 Millisecond Duration
Lung Weight, Percent of Body Weight
2.5
2.0
1.5
1.0
0.5
n=75 Controls
n=10
n=25
n=32
n=3
n=13
n=17
n=5
n=15
n=4
n=21
n=1
n=14
Pressure Group
Pressure
1
18.6
2
30.8
3
36.4
4
38.0
5
39.3
6
41.8
7
46.5
Reflected Pressure, psi

Figure 8

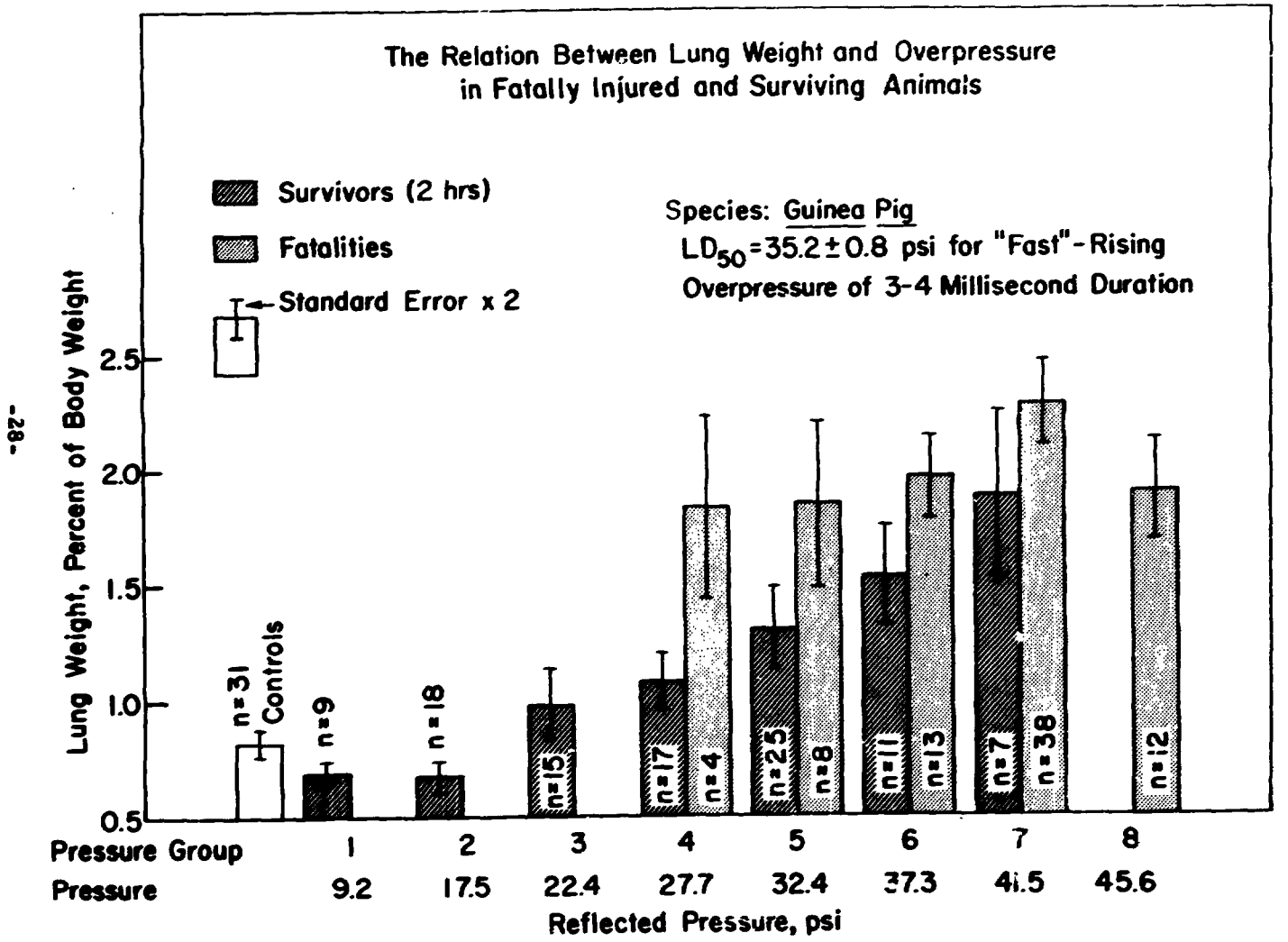

The Relation Between Lung Weight and Overpressure
in Fatally Injured and Surviving Animals
Survivors (2 hrs)
Fatalities
Standard Error x 2
Species: Guinea Pig
LD50=35.2±0.8 psi for "Fast"-Rising
Overpressure of 3-4 Millisecond Duration
Lung Weight, Percent of Body Weight
2.5
2.0
1.5
1.0
0.5
n=31 Controls
n=9
n=18
n=15
n=17
n=4
n=25
n=8
n=11
n=13
n=7
n=38
n=12
Pressure Group 1 2 3 4 5 6 7 8
Pressure 9.2 17.5 22.4 27.7 32.4 37.3 41.5 45.6
Reflected Pressure, psi

Figure 9

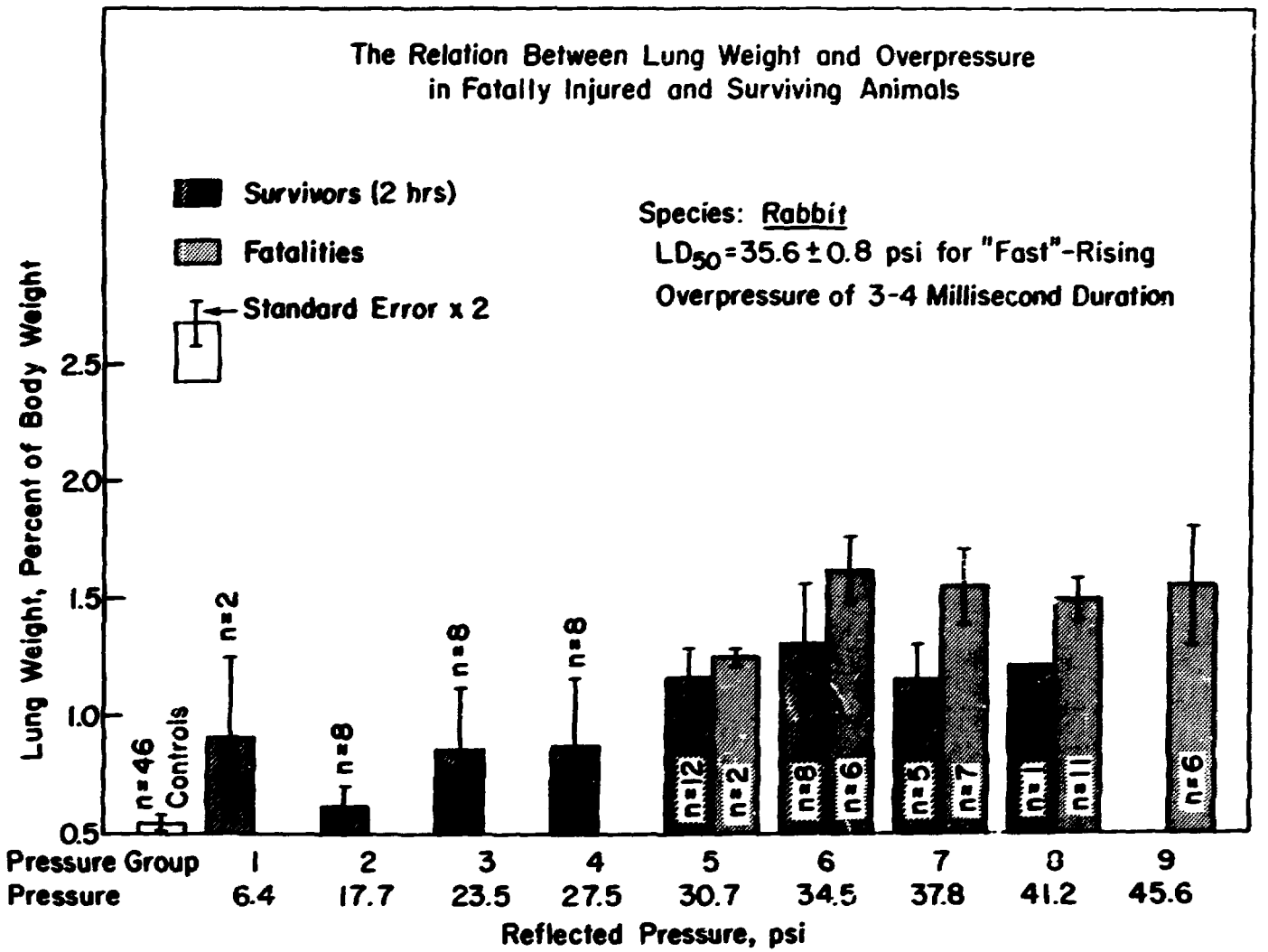

The Relation Between Lung Weight and Overpressure
in Fatally Injured and Surviving Animals
Survivors (2 hrs)
Fatalities
Standard Error x 2
Species: Rabbit
LD50=35.6±0.8 psi for "Fast"-Rising
Overpressure of 3-4 Millisecond Duration
Lung Weight, Percent of Body Weight
0.5
1.0
1.5
2.0
2.5
n=46 Controls
n=2
n=8
n=8
n=8
n=12
n=2
n=8
n=6
n=3
n=7
n=1
n=11
n=6
Pressure Group 1 2 3 4 5 6 7 8 9
Pressure 6.4 17.7 23.5 27.5 30.7 34.5 37.8 41.2 45.6
Reflected Pressure, psi

Figure 10

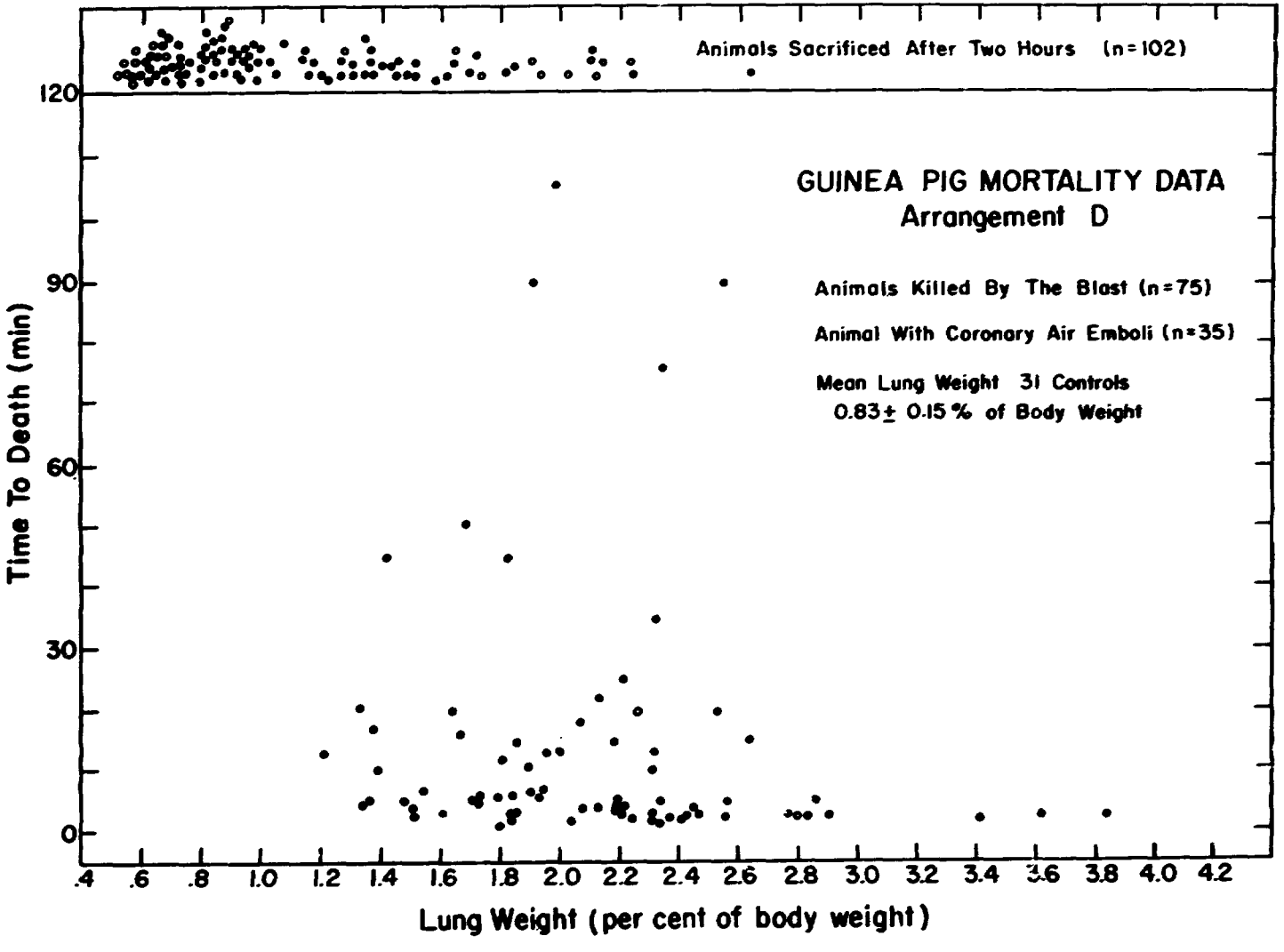

Animals Sacrificed After Two Hours (n=102)
GUINEA PIG MORTALITY DATA
Arrangement D
Animals Killed By The Blast (n=75)
Animal With Coronary Air Emboli (n=35)
Mean Lung Weight 31 Controls
0.83± 0.15 % of Body Weight
Time To Death (min)
120
90
60
30
0
.4 .6 .8 1.0 1.2 1.4 1.6 1.8 2.0 2.2 2.4 2.6 2.8 3.0 3.2 3.4 3.6 3.8 4.0 4.2
Lung Weight (per cent of body weight)

Figure 11

a threshold in terms of overpressure to be established for lung hemorrhage, it can be said that the finding was quite prominent as low as about 7 psi for the mouse and between 17 and 18 psi for the other three species.

Arterial air embolism. The numbers of fatally blasted guinea pigs and rabbits in which coronary and cerebral arterial air emboli were visualized during post mortem examination are listed in Tables 13 and 14, grouped to show the relation to overpressure. Air emboli were noted in the coronary arteries of 47 per cent and 22 per cent of fatally wounded guinea pigs and rabbits, respectively, whereas air emboli were seen in the cerebral arteries in 41 and 31 per cent of the two species, respectively.

Coronary air emboli were not visualized in any of the surviving animals nor in those who died after 10 min. Figure 11 shows the individual occurrence of coronary air emboli for the guinea pig as a function of time. It is evident from the figure that the incidence of air emboli in mortally injured animals increases with increasing lung weight and is very high in animals dying within the first and second 5-minute periods.

Coronary and cerebral air embolism were not assessed in mice and rats in this study; however, in subsequent experiments arterial air emboli were observed in the coronary arteries of both species with the aid of optical equipment.

Hemothorax and pneumothorax. The occurrence of hemothorax and pneumothorax in the four species of animals studied is summarized in Tables 15 through 18. The data were grouped to show these signs in surviving and fatally wounded animals as they varied with overpressure. Except for the guinea pig, hemothorax was a common lesion produced by the blast especially in fatalities associated with exposure to the higher overpressures. On the average — again excepting the guinea pig — there was a highly significant statistical difference between the incidence of hemothorax in survivors and in mortally injured animals, as noted in the probability figures in Tables 15 through 18. The figures were obtained using the chi square test[7] and apply to the total figures shown. With regard to the exceptional findings of a low incidence of hemothorax in the guinea pig, it may be significant that this species — compared with the rabbit at least — showed a high average incidence of coronary

Table 13

The Incidence of Arterial Air Embolism in Guinea Pigs Killed by the Blast*

Group number	Reflected pressure psi	Number of deaths	Number with coronary air emboli	Number with cerebral air emboli
1	9.2	0	-	-
2	17.5	0	-	-
3	22.4	0	-	-
4	27.7	4	0	0
5	32.4	8	4(50%)	3(38%)
6	37.3	13	3(23%)	3(23%)
7	41.5	38	22(60%)	18(47%)
8	45.6	12	6(50%)	7(58%)
Totals		75	35(47%)	31(41%)

*Air emboli were not found in the circulatory system of any of the guinea pigs that survived the two hour postshot period.

Table 14

The Incidence of Arterial Air Embolism in Rabbits Killed by the Blast*

Group number	Reflected pressure psi	Number of deaths	Number with coronary air emboli	Number with cerebral air emboli
1	6.4	0	-	-
2	17.7	0	-	-
3	23.5	0	-	-
4	27.5	0	-	-
5	30.7	2	0	0
6	34.5	6	0	0
7	37.8	7	2(29%)	2(29%)
8	41.2	11	4(36%)	6(55%)
9	45.6	6	1(17%)	2(33%)
Totals		32	7(22%)	10(31%)

*Air emboli were not found in the circulatory system of any of the rabbits that survived the two hour postshot period.

Table 15

The Occurrence of Hemothorax and Pneumothorax in the Mouse

Group numbers	Reflected shock pressure psi	Survivors			Fatalities		
		Number of cases	No. with hemo-thorax	No. with pneumo-thorax	Number of cases	No. with hemo-thorax	No. with pneumo-thorax
1	7.6	30	0	0	0	--	--
2	16.2	30	1	0	0	--	--
3	22.8	37	6	1	3	2	0
4	24.8	18	5	1	2	0	0
5	29.7*	11	3(27%)	0	19	3(16%)	0
6	35.5	0	-	-	20	8	1
7	39.1	3	1	0	27	18	6
8	40.3	1	1	0	29	20	3
9	45.5	0	-	-	10	5	1
Totals**		130	17(13%)	2(1.5%)	110	56(51%)	11(10%)

*Computed LD_{50} = 29.0 psi.

**Probability of significant difference: Hemothorax, $<<0.001$; Pneumothorax, 0.01 - 0.001.

Table 16

The Occurrence of Hemothorax and Pneumothorax in the Rat

Group numbers	Reflected shock pressure psi	Survivors			Fatalities		
		Number of cases	No. with hemo-thorax	No. with pneumo-thorax	Number of cases	No. with hemo-thorax	No. with pneumo-thorax
1	18.6	10	0	0	0	-	-
2	30.8	25	0	0	0	-	-
3	36.4	32	7	0	3	1	0
4	38.0*	13	4(30%)	0	17	5(29%)	0
5	39.3	5	0	0	15	1	0
6	41.8	4	0	0	21	9	3
7	46.5	1	1	0	14	6	1
Totals**		90	12(13%)	0	70	22(31%)	4(6%)

*Computed LD_{50} = 38.6 psi

**Probability of significant difference: Hemothorax, 0.01 - 0.001; Pneumothorax, 0.05 - 0.02.

Table 17

The Occurrence of Hemothorax and Pneumothorax in the Guinea Pig

Group numbers	Reflected shock pressure psi	Survivors			Fatalities		
		Number of cases	No. with hemo-thorax	No. with pneumo-thorax	Number of cases	No. with hemo-thorax	No. with pneumo-thorax
1	9.2	9	0	0	0	-	-
2	17.5	18	0	0	0	-	-
3	22.4	15	0	0	0	-	-
4	27.7	17	0	0	4	0	0
5	32.4	25	0	0	8	0	0
6	37.3*	11	0	0	13	0	0
7	41.5	7	1	0	38	2	0
8	45.6	0	-	-	12	0	0
Totals**		102	1(1%)	0	75	2(3%)	0

*Computed LD_{50} = 35.2 psi

**Probability of significant difference: Hemothorax, 0.5 - 0.3.

Table 18

The Occurrence of Hemothorax and Pneumothorax in the Rabbit

Group numbers	Reflected shock pressure psi	Survivors			Fatalities		
		Number of cases	No. with hemo-thorax	No. with pneumo-thorax	Number of cases	No. with hemo-thorax	No. with pneumo-thorax
1	6.4	2	0	0	0	-	-
2	17.7	8	0	0	0	-	-
3	23.5	8	0	0	0	-	-
4	27.4	8	0	0	0	-	-
5	30.7	12	0	0	2	1	1
6	34.5*	8	1(13%)	1(13%)	6	1(17%)	0
7	37.8	5	0	0	7	3	2
8	41.2	1	0	0	11	5	3
9	45.6	0	-	-	6	3	1
Totals**		52	1(2%)	1(2%)	32	13(40%)	7(22%)

*Computed LD_{50} = 35.6 psi

**Probability of significant difference: Hemothorax, $<<0.001$; Pneumothorax, 0.01 - 0.001.

air embolism, as noted in Tables 13 and 14. Over-all, 47 and 22 per cent of guinea pig and rabbit fatalities, respectively, exhibited coronary air emboli. In some of these cases heart failure could have occurred very early. This event would tend to lower the blood pressure and perhaps minimise the occurrence of hemothorax; certainly, it would sharply curtail lung hemorrhage and, therefore, the association of low lung weights with fatality would be expected in some cases. That this indeed occurred was noted above and is documented for the guinea pig in Fig. 11.

In contrast to hemothorax pneumothorax was a relatively infrequent finding in fatalities and was very rare in 2-hr survivors. Tables 15 through 18 show that only 1.5 (2 animals) and 2.0 (1 animal) per cent of surviving mice and rabbits, respectively, exhibited pneumothorax, whereas only 10, 6, and 22 per cent of mortally injured mice, rats, and rabbits, respectively, showed this sign. Pneumothorax was not noted in any of the exposed guinea pigs.

In spite of the low incidence of pneumothorax, there was, on the average, a significant difference between fatally wounded and surviving animals. The probabilities were much less than one in a hundred that the differences noted in mice and rabbits were due to chance alone. In the case of the rat, the probabilities were less than five in a hundred that the observed occurrence of pneumothorax among survivors and fatalities was significantly different. (See Tables 15 through 18.)

Intra-abdominal lesions. Major injuries to the abdominal viscera noted during post-mortem examination involved mainly the spleen and the gastrointestinal tract. These lesions commonly consisted of subcapsular contusions of the spleen and hemorrhagic areas in the lining of the stomach and large intestine. Yet rarer, and associated usually with exposure to the higher overpressures, were rupture of these organs. Hemoperitoneum was noted in cases of ruptured organs, but on two occasions the source of intraperitoneal bleeding was not established.

The incidence of these abdominal signs for guinea pigs and rabbits is summarized in Tables 19 and 20, respectively. The data were arranged to allow comparison of animals surviving 2 hr with those killed by the blast and to show the relation to overpressure.

Table 19

The Occurrence of Intra-abdominal Injuries in Guinea Pigs

Pressure group	Survivors				Fatalities			
	n	Spleen contused	Stomach hemorrhage	Intestinal hemorrhage	n	Spleen contused	Stomach hemorrhage	Intestinal hemorrhage
1 (9.2)	9	0	0	0	0	-	-	-
2 (17.5)	18	0	10	4	0	-	-	-
3 (22.4)	15	2 (2)*	7	12	0	-	-	-
4 (27.7)	17	5 (1)	14	7	4	0	2	3
5 (32.4)	25	3 (2)	20	23	8	4 (4)*	8	8
		11%**	81%	89%		38%	81%	100%
6 (37.3)	11	1 (1)	9	9	13	4	9	13 (1)
7 (41.5)	7	4	7	7	38	24 (8)	35 (2)*	38
8 (45.6)	0	-	-	-	12	12 (7)	12 (1)	12
Totals†	102	15 (14.7%)	67 (65.7%)	62 (60.8%)	75	44 (58.7%)	66 (88.0%)	74 (98.7%)

*The number of cases with rupture of the indicated organ enclosed in parentheses.

**The percentage incidence obtained when groups 5 and 6 were combined (computed LD_{50} = 35.2 psi).

†Probability of significant difference: Spleen, $<<0.001$; Stomach, <0.001; Intestine, $<<0.001$.

Table 20

The Occurrence of Intra-abdominal Injuries in Rabbits

Pressure group	Survivors n	Spleen contused	Stomach hemorrhage	Intestinal hemorrhage	Fatalities n	Spleen contused	Stomach hemorrhage	Intestinal hemorrhage
1 (6.4)	2	0	0	0	0	-	-	-
2 (17.7)	8	0	1	1	0	-	-	-
3 (23.5)	8	0	5	2	0	-	-	-
4 (27.4)	8	0	6	3	0	-	-	-
5 (30.7)	12	0	5	8 (1)*	2	0	1	1
6 (34.5)	8	0	3	5 (1)	6	1	1	3 (1)
		8%**	62%	62%		23%	46%	69%
7 (37.8)	5	1	5	3	7	2	5	6 (2)
8 (41.2)	1	0	1	1	11	3	6	9 (1)
9 (45.6)	0	-	-	-	6	2	2	5 (1)
Totals†	52	1 (1.9%)	26 (50.0%)	23 (44.2%)	32	8 (25.0%)	15 (46.9%)	24 (75.0%)

*The number of cases with rupture of the indicated organ enclosed in parentheses.

**The percentage incidence obtained when groups 6 and 7 were combined (computed LD_{50} = 35.6 psi).

†Probability of significant difference: Spleen, <0.001; Stomach, 0.80 - 0.70; Intestine, 0.01 - 0.001.

In the case of the guinea pig, contusion of the spleen and hemorrhage of the mucosa of the stomach and intestines were prominent signs in both surviving and mortally wounded animals. Though the total figures showed a highly significant difference between the two groups, the probability being much less than one in one thousand that chance alone was responsible, it is true that this difference was much less marked and only significant ($p = 0.01\text{-}0.02$) for contused spleens when the comparison was made for animal exposure near the LD_{50} overpressure, as shown by the percentage figures near the center of Table 19. This probably means that the abdominal signs were not critical with regard to early mortality, though they could have been contributing factors. The latter might be particularly true with regard to splenic rupture which occurred in only 3 of 36 surviving animals exposed near the LD_{50} pressure, but in 4 of 21 fatally injured animals subjected to the same overpressure. Over-all, however, there were 6 and 19 ruptured spleens observed in surviving and "fatal" groups, respectively. Even less frequent were three ruptures of the stomach and one rupture of the large intestine seen in mortally wounded guinea pigs only.

The rabbit, as shown in Table 20, exhibited fewer abdominal signs than did the guinea pig. Even so, except for hemorrhage of the stomach mucosa, there was an average difference that was quite significant between the incidence of splenic contusion and mucosal hemorrhage of the intestine as recorded in the surviving and fatally injured groups. Comparison near the LD_{50} overpressure, as was the case with the guinea pigs, showed less marked differences between dying and surviving animals. Too, there were only two and five ruptured large intestines in the latter and former groups, respectively.

In contrast to the guinea pigs and rabbits, only 9 rats and no mice among all animals exposed sustained mucosal hemorrhage of the gastrointestinal tract. Lesions of the stomach and spleen were remarkable by their absence in rats and mice, as was also the case for liver damage in all four species of animals.

Eardrum rupture. Though the eardrum data will be presented elsewhere it is well to note here that except for the lowest pressure groups included in this study, the magnitudes of the overpressures were well above those necessary to rupture all the eardrums of the four animal species.[12] For example, there was 100 per cent rupture of the tympanic membrane in all guinea pigs above pressure group 1 (see Table 4). In group 1 the range of maximal overpressure was

from 9.1 to 9.5 psi and perforation of the eardrum occurred in 95 per cent of the ears exposed.

DISCUSSION

General

By way of discussion and to amplify the meaning of the data reported above, attention will be directed first to the results of other recent experiments which, like those reported here were designed primarily to establish the relation between interspecies mortality and single-pulse, "fast"-rising overpressures of various magnitudes and durations. Second, significant and interesting observations incidentally made during such studies will be noted wherein they bear mostly upon the acute biological consequences of exposure. Too, pertinent and related information from the literature will be cited.

The Overpressure-Duration Relationship

To date – including the present study – three series of experiments, each involving similar instrumentation and sizable numbers of animals, have been completed in investigations of the biological significance of the pressure-duration relationship for single-pulse, "fast"-rising overpressures. The overpressure durations employed covered a wide range and were 6-8 sec,[11] 400 msec,[13] and 3-4 msec (the present study); mice, rats, guinea pigs, and rabbits were used plus dogs and goats for the 400 msec series; all species exposed to blast, except the dogs and goats for which a harness was used, were located in cages bolted against the end-plate of a suitably designed and instrumented shock tube. The LD_{50} data are summarized in Table 21 which also details the number of animals in each experimental series.

A study of the table will convince the reader that the maximal overpressures required to produce 50 per cent acute mortality in mice, rats, guinea pigs, and rabbits were essentially the same for each individual species even though the overpressure durations ranged from 3-4 msec to 6-8 sec

That this might not be true for shorter duration overpressures and for larger animals was alluded to in the introduction. Specifically, Fisher et al.[8] reported LD_{50} figures for rabbits, monkeys, and goats near 55, 94, and 200 psi, respectively, using small high-explosive charges and, therefore, "short"-

Table 21

Maximal Single Pulse "Sharp"-rising Overpressures of Indicated Duration Required for 50 Per Cent Mortality

Species of animal	Maximal overpressures of indicated durations producing a mortality of 50 per cent						Total number animals
	6-8 sec*		400 msec**		3-4 msec**		
	No. animals	LD_{50}† psi	No. animals	LD_{50}† psi	No. animals	LD_{50}† psi	
Mouse	115	29.8±1.1	140	30.9±0.5	240	29.0±0.6	495
Rat	55	38.7±0.6	164	36.3±0.5	160	38.6±0.8	379
Guinea pig	140	36.7±0.7	96	35.2±0.6	177	35.2±0.8	413
Rabbit	145	33.4±1.2	104	32.4±1.3	84	35.6±0.8	333
Dog			35	50.7±1.2			35
Goat			30	53.0±2.8			30
Totals	455		569		661		1685

*Lethality in 1 hr.
**Lethality in 2 hrs.
†The plus or minus figures represent the standard error.

duration overpressures. Benzinger[1] and Schardin[16] also cited a figure over 50 psi for the lethal limit for guinea pigs, again applying to small high-explosive charges. These data all indicate that for small enough charges and thus, short enough durations, the overpressure required for a given mortality increases sharply.

The same statement apparently applies qualitatively to larger animals as the findings of Desaga for dogs[5] noted in the introduction indicate. However and quantitatively for the larger animal, the overpressure duration shorter than which the overpressure for lethality rises markedly is longer than it is for smaller animals. This fact is consistent with results for animals the size of cows as Desaga has reported[5] in high-explosive studies employing charges of 4400 lbs.

Recent, but as yet incomplete, carefully instrumented experiments in Albuquerque using high-explosive charges from a few ounces to 64 lbs has confirmed the earlier findings cited above. Figure 12 graphically summarizes these and similar data from the literature. The latter information was selected to include only experiments for which both overpressure and duration were either measured or could be scaled with reasonable reliability from published information. The figure shows the lethality curves for "large" and "small" animals as a function of maximal overpressure and pulse duration. Though it is apparent the data are as yet somewhat fragmentary, particularly for the larger animals, it seems clear that: first and generally, for "sharp"-rising overpressures there is a "critical" pulse duration shorter than which the overpressure for mortality rises rapidly and thus, both overpressure and pulse duration are definitive for lethality; second and also generally, for overpressures longer than the critical duration, it is only the magnitude of the overpressure that is significant for lethality or damage; third and more specifically, the critical duration varies with animal size, being on the order of many hundreds of microseconds to a very few milliseconds for smaller animals and a few to many tens of milliseconds for larger animals.

A more precise understanding of the pressure-duration relationship must await additional data to better define the critical duration for each species and to establish the shape of the pressure-duration curves for minor and serious injury as they apply to mammals below and above the weight of man. Similarly,

Lethality Curves for "Larger" and "Smaller" Animals

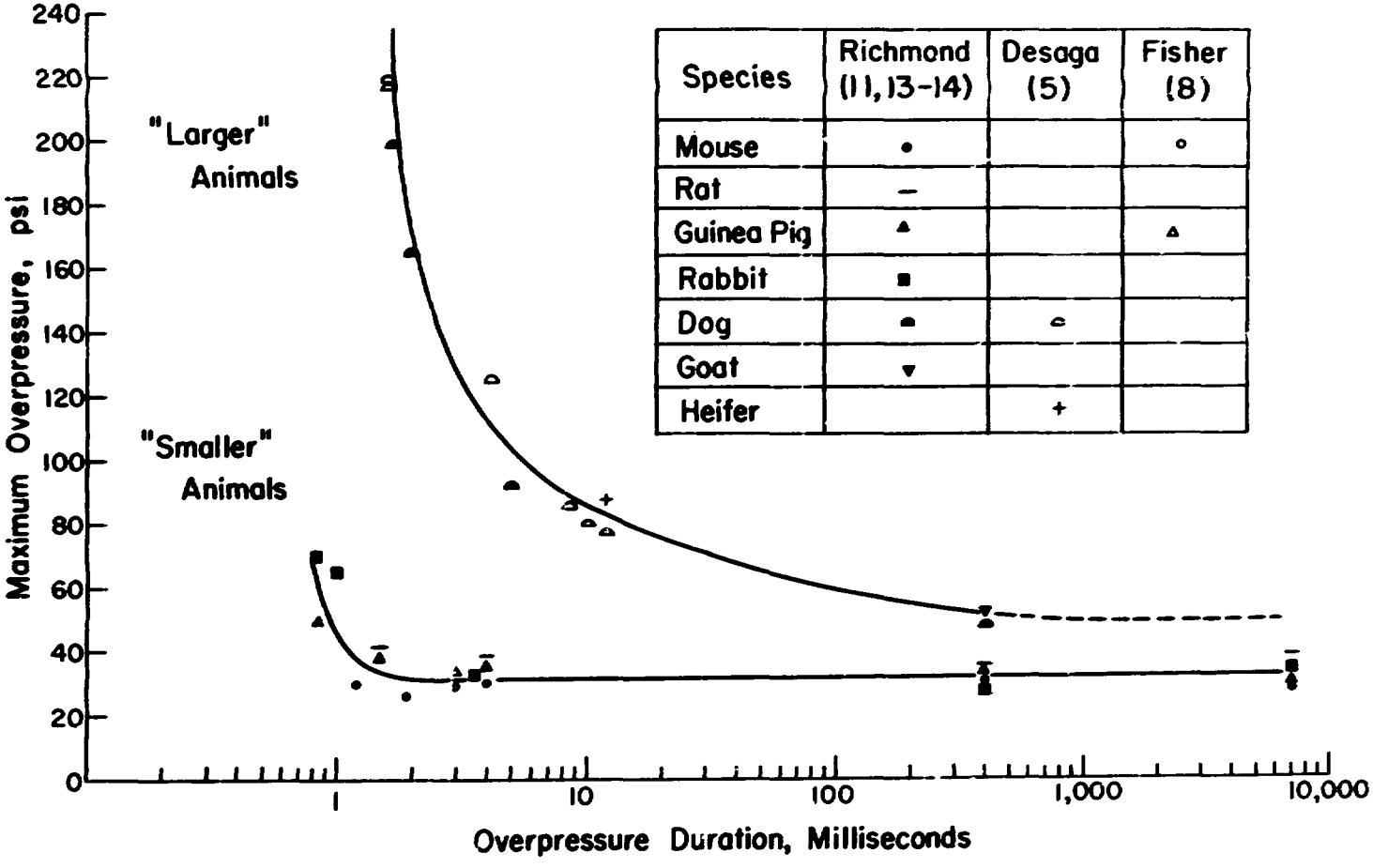


Figure 12

tolerance for the very young and very old yet remains to be investigated if the predictions for the human case noted in the introduction are to be further refined.

The interspecies data presented in Fig. 12 reveals one other point of interest; namely, the close correspondence between the mortality figures obtained with the shock tube on the one hand and high-explosive charges on the other. It is significant that the shock-tube data represent maximal pressures which resulted from the reflection of the incident pressure against the end-plate of the tube. Thus, the animal was exposed to the incident pressure and its reflection almost instantaneously. The recent high-explosive experiments in Albuquerque, giving quantitatively similar results, also were arranged to allow exposure of the animals almost instantaneously to the incident pressure and its reflection; e.g., animals were located on a concrete pad beneath the exploded charge. To the contrary, animals exposed on the ground to blast from high-explosive charges detonated on the ground are subjected to a maximal pressure which is equivalent only to the incident pressure Again, similar results are obtained as shown by Fig. 12. Thus, all other factors including pulse duration being equal, it is the maximal overpressure that is important and it matters not whether this is the incident or the incident plus the reflected pressure, providing the last two are applied almost instantaneously. However, if the rising phase of the overpressure is slow[10] or occurs in two steps, as it does if an animal is mounted at various distances from a reflecting surface,[11, 20] a circumstance which allows appreciable time between the application of the incident and the reflected pressure, then much greater maximal pressures are required for lethality.

These facts are important in estimating biological blast effects as a function of explosive yield and range; e.g., if circumstances allow exposure to sharply-increasing, maximal reflected pressures, the range for a given effect would be much greater than would be the case if exposure only to the maximal incident pressure were involved.

The Mortality-Overpressure Relationship

There are at least three points of interest concerning the present and other similar studies, which are related to the observed relationship between the magnitude of the overpressure and lethality. The first is the fact that the probit analy-

sis of Finney,[6] a technique for linearizing "S"-shaped mortality curves, was used to analyze the data. Since this technique presents mortality in units which are multiples of the standard deviation above and below the 50 per cent mortality point as a function of the log of the maximal overpressure, it is well to realize that Fig. 5 presents a statistically "weighted" situation in which the data points closest to the 50 per cent mortality have greater importance than those with either very high or very low mortality. One reflection of this is the "artificial" widening of the 95 per cent confidence lines at the higher and lower ends of the mortality scales.

Second, and related to the above remarks, is the fact that among the several species of animals there was a spurious variation in the groupings of pressure and hence in the data points for each species; e.g., sometimes there were two points above and below the 50 per cent mortality value as with the rabbit; sometimes there were four points above the probit value of 5 and one below as was the case with the rat; and sometimes the pressure groups were not symmetrically located about the 50 per cent value. Relevant to the discussion is the steepness of the probit mortality lines; i.e., because there is not much variation in pressure between low and high mortality along with the innate variability in biologic response to pressure variation, it is difficult to choose and achieve an ideal distribution of data points. Experience, however, has shown that these spurious factors do not influence the LD_{50} values very much, as can be seen by studying Table 21. However, the opposite is true as far as the slopes of the probit regression equations are concerned; i.e., these values are much more sensitive to data points at the high and low end of the mortality range, as a study of Table 6 and Fig. 5 will show, particularly in the case of the rat, the species in which the slope of the regression line was quite different than for the other three species.

Third, and also related to the spurious variation in the pressure groups as they might influence the analytical treatment of the data, is the time at which mortality is assessed. This is important because, on the average, animals exposed to overpressures well above the LD_{50} values tend to die sooner than those exposed to pressures which are well below the 50 per cent lethality figure. This means that the time factor might influence not only the slope constant, but also the LD_{50} figures as well. That the latter indeed occurs

is illustrated by Table 22 summarizing data from a recent study[7] showing the difference in LD_{50} values when mortality was arbitrarily assessed at 1 and 24 hours, as well as at 2 hours as noted in the 400 msec values presented in Table 21. Attention is directed to the rabbit figures in Table 22 showing a change in the LD_{50} values from 32.7 psi for 1 hr mortality to 29.6 psi for 24 hr mortality. A similar drop in the LD_{50} figures is also apparent for the dog.

In spite of the variability introduced by the time factor just mentioned and the spurious location of data points about the LD_{50} values, there is a surprising stability in the results obtained to date for the several species as a study of Tables 21 and 22 will show. Even so, considerable care in data analysis is required and it remains for the future to confirm or deny that (a) the slopes of the probit curves are in reality fairly constant among the several species, and (b) the LD_{50} values are significantly different than those now available when mortality studies are carried on over a matter of days and weeks instead of hours.

Time of Death

The early death of mortally wounded animals, shown in Table 7 and Fig. 6 and noted in previous studies as characteristic of severe injury from primary blast[20] and violent impact,[15] deserves considerable emphasis for several reasons. First, the cumulative mortality-time curves of Fig. 6 are remarkable in that they followed about the same pattern for each species, which fact strongly suggests that all animals died of a common blast syndrome.

Second, the mortality-time curves tentatively can be viewed as helpful in assessing the probable cause of death. For example, the initial steep portion of the curves covering the first 5 to 10 min might well be due to either massive hemorrhage or massive arterial air emboli involving the heart and/or the central nervous system. The following less steep portion of the curves could indicate death from suffocation, if hemorrhage continued or bloody froth obstructed the airways, or from progressive embarrassment of the heart by air emboli. The final and flatter portion of the curve might involve cardiac and pulmonary embarrassment due to the development of pulmonary edema, poor oxygenation of the blood, elevation of pulmonary and venous pressures with consequent failure

Table 22

Variation in the Maximal "Sharp"-rising Overpressures of 400 Msec Duration Required for 50 Per Cent Lethality When Mortality is Assessed at 1 Hour, 2 Hours, and 24 Hours

	Maximal overpressure (incident or reflected) required for 50 per cent mortality at the indicated times - in psi		
Species	1 hour	2 hours	24 hours
Mouse	31.4 ± 0.5	30.9 ± 0.5	30.7 ± 0.6
Rat	36.6 ± 0.5	36.3 ± 0.6	36.3 ± 0.6
Guinea pig	35.4 ± 0.6	35.2 ± 0.6	34.5 ± 0.6
Rabbit	32.7 ± 1.4	32.4 ± 1.3	29.6 ± 0.9
Dog	52.2 ± 1.9	50.7 ± 1.2	47.8 ± 1.1
Goat	53.0 ± 2.8	53.0 ± 2.8	53.0 ± 2.8

of the right heart, and a critical fall in the systemic arterial pressure producing a shock-like picture ending eventually in fatality.

Third, the rapidity with which primary blast death ensues also has other important clinical implications. Certainly, any therapy to be effective in very many cases must be instigated in a matter of very few tens of minutes after injury. This emphasizes the need for planned rescue operations by trained medical teams in cases of accidental explosions, say at powder plants or transportation centers handling explosives. Likewise, the need for prophylactic measures to avoid or minimize primary blast injury is brought into sharp focus and this statement has significance in the context of nuclear war as well as test and routine; commerical manufacturing operations.

Miscellaneous

Nasal and oral signs. It is well to point out here that in other studies in Albuquerque[11, 13-14] the appearance of bloody froth and/or hemorrhage from the nose and mouth has been observed in animals surviving exposure to blast. Consequently, the absence of these signs in all survivors of the present study shown in Table 8 should not be taken as applicable to all blast experience. However, it is clear that such findings in any case of blast injury – and probably of thoracic trauma – should alert the physician to the probable existence of severe and dangerous damage to the lung.

Experimental design. The comparisons, given in the results section, of the incidence of various signs and symptoms in survivors and dying animals deserves some comment. Since the current study was designed to establish mortality-pressure relationships for "sharp"-rising overpressures of 3-4 msec duration, the animals surviving and expiring were few in number at the "high" and "low" ends of the pressure scale, respectively. Thus, the comparison of symptoms among mortally injured and surviving cases suffered because often small samples had to be compared with large ones. It is clear that a careful attack on symptomalogy, wherein definitions of signs and symptoms critical for mortality were desired, would require specially designed experiments. The ideal course would be to work very close to the 50 per cent killing pressure for each species. Thereby equal numbers of living and dying animals would be available and the chances of obtaining the best comparative data would be much enhanced.

Combined injury. Also related to the design of the experiments reported here is the fact that all animals were exposed in a manner to minimize displacement on the one hand and eliminate blast energized missiles on the other hand. Thereby the biological effects, mostly due to pressure variations alone, were studied. Animals not so restrained would have been subjected not only to the additional trauma of differential displacement of different portions of the body, but also to gross bodily displacement which might prove damaging during the accelerative and/or the decelerative phase of such experience. Because of these facts, the reader should realize that grave and even fatal blast injury, particularly from missile or violent impact, might well be associated with much lower overpressures than reported here. Too, it is possible that the threshold for pressure effects might be lowered by the occurrence of other blast-produced trauma. However, it is likewise realistic to point out that real-life exposures to blast may occur in situations which minimize missile production and displacement. This certainly has occurred by chance in explosive accidents and by plan when protective structures have been utilized to minimize blast injury and fatality. Under such circumstances — trauma from displacement and missiles being minimized — the effects of overpressure alone can be the important and critical hazard.

SUMMARY

1. Four species of animals were exposed, one in a cage, to shock tube-produced, "fast"-rising overpressures of 3-4 msec duration primarily to determine the relationship between mortality and the magnitude of the overpressure.

2. The maximal overpressures associated with 50 per cent mortality were 29.0, 38.6, 35.2 and 35.6 psi for mice, rats, guinea pigs and rabbits, respectively.

3. Times of death for mortally wounded animals were determined over a 120 minute period; death occurred rapidly, the figures showing 65, 93, 96 and 100 per cent fatalities within 5, 30, 60 and 120 minutes, respectively. That such facts highlighted the need for early therapy and prophylactic protective measures was pointed out and emphasized.

4. The appearance of blood-tinged froth and/or frank, but transient hemorrhage at the nares and mouth was observed in 63 per cent of fatally injured ani-

mals. These grave signs were seen in no animal surviving blast exposure in this study though it has been seen in other experiments.

5. Characteristic lung hemorrhage was the most serious lesion noted and the severity of the lesion was, on the average, correlated with the magnitude of the overpressure.

6. Lung hemorrhage was observed in non-fatal exposures to overpressures as low as 7 psi for mice and between 17 and 18 psi for rats, guinea pigs and rabbits.

7. An increase in lung weight expressed as a per cent of body weight was significantly higher, on the average, in fatalities than in survivors. The greatest variability in lung weight was observed in animals expiring within 5 min. The increase of lung weight in fatal cases frequently was two- and three-fold that of controls.

8. Arterial air embolism was observed in the coronary and cerebral vessels of guinea pigs and rabbits, but not in mice and rats (though in subsequent studies the latter two species exhibited these signs). Coronary air emboli, on the average, appeared in 47 and 22 per cent of expiring guinea pigs and rabbits, respectively, while the corresponding figures for cerebral emboli were 41 and 31 per cent.

9. Coronary air emboli in guinea pigs were visualized in animals expiring within 5 and 10 min, but not in animals dying later; neither were air emboli seen in any animal sacrificed after 2 hrs.

10. The incidence of hemothorax, pneumothorax and intra-abdominal lesions was tabulated in mortally wounded and surviving animals. Hemothorax was a common lesion in all expiring animals except the guinea pig. Pneumothorax, in contrast, was a relatively infrequent lesion being very rare in survivors.

11. The average incidence of hemothorax was 51, 31, 3 and 40 per cent in fatally wounded mice, rats, guinea pigs and rabbits, respectively, and except for the guinea pig, this lesion was significantly more prominent than in survivors.

12. Pneumothorax was a relatively rare finding, showing an average in-

cidence of 10, 6, 0 and 22 per cent in expiring mice, rats, guinea pigs and rabbits, respectively. In survivors, only 2 and 1.5 per cent of rabbits and mice, respectively, showed this lesion.

13. Intra-abdominal damage in guinea pigs and rabbits included mucosal hemorrhage of the stomach and intestines, contusion and rupture of the spleen and rupture of the stomach and intestines. On the average, all abdominal signs were significantly higher in fatal than in surviving cases except for mucosal hemorrhage of the stomach mucosa in rabbits.

14. Selected data from the literature were reviewed and compared with the present and other recent studies in Albuquerque wherein many hundreds of animals have been exposed to single-pulse, "sharp"-rising overpressures varying in duration from less than 1 millisecond up to 6-8 seconds.

15. A critical pulse duration, shorter than which the overpressure for mortality rises sharply and beyond which only the magnitude of the overpressure is definitive for mortality, is apparent for each animal species.

16. The critical pulse duration varies with animal size and is on the order of many hundreds of microseconds to very few milliseconds for smaller animals, (mice, rats, guinea pigs and rabbits) and a few to many tens of milliseconds for larger animals (dogs, goats and cows).

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